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REVIEW ARTICLE

Early prediction of femoral head avascular necrosis following neck fracture

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Summary Femoral neck fracture puts at risk functional prognosis in young patients and can be life-threatening in the elderly. The present study reviews methods of femoral head vascularity assessment following neck fracture, to address the following issues: what is the risk of osteonecrosis? And what, in the light of this risk, is the best-adapted treatment to avoid iterative surgery? Femoral head vascularity depends on retinacular vessels and especially the lateral epiphyseal artery, which contributes from 70 to 80% of the femoral head vascular supply. Fracture causes vascular lesions, which are in turn the prime cause of necrosis. Other factors combine with this: hematoma tamponade effect, reduced joint space and increased pressure due to lower extremity positioning in extension/internal rotation/abduction during surgery. Head deformity is not due to direct cell death but to the repair process originating from the surrounding living bone. In post-traumatic necrosis, proliferation rapidly invades the head, with significant osteogenesis. Pathologic fractures occur at the boundary between the new and dead bone. Many techniques have been reported to help assess residual hemodynamics and risk of necrosis. Some are invasive: superselective angiography, intra-osseous oxygen pressure measurement, or Doppler-laser hemodynamic measurement; others involve imaging: scintigraphy, conventionnal or dynamic MRI. The future seems to lie with dynamic MRI, which allows a new classification of femoral neck fractures, based on a non-invasive assessment of femoral head vascularity.

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Introduction

Femoral neck fracture is a serious lesion, life-threatening in the elderly and threatening functional prognosis in the young. It is a frequent pathology, with an annual incidence of some 1/1000 of the population [1]. The vascular impact is 3-fold: displacement, interrupting retinacular vessels [2]; rotation or valgus, interrupting ligament teres vascularization [3]; and increased intra-capsular pressure, producing a tamponade effect [4]. The basic anatomic risk is post-traumatic osteonecrosis, estimated at 10 to 30% [5,6]. In elderly patients, this is life threatening, with a mortality rate of 20 to 30% in the year following the fracture [1], and treatment aims at a rapid restoration of the patient's autonomy. Given the risk of head necrosis and the poor fixation of osteosynthesis material in osteoporotic bone, conservative treatment is difficult in displaced fracture (Garden III–IV), but may be attempted in low-grade displacement or stable fracture (Garden I–II) although the risk of necrosis is not to be forgotten. In young patients, treatment should be conservative, but the functional prognosis basically depends on whether necrosis occurs; to limit this risk, osteosynthesis should be performed as early as possible, with anatomic reduction and stable assembly [1].

The present study reviews the options for early (within the first hours following fracture) assessment of residual head perfusion following neck fracture as an index of necrosis risk. The aim is not to give a technical description of the techniques so much as to enable management to be optimised and iterative surgery to be avoided, by addressing the questions the traumatologist has to answer in case of recent femoral neck fracture: how significant is the risk of necrosis, and which form of treatment is best adapted in the light of that risk?

Normal femoral head vascularization

Many studies of femoral head vascularization have assessed lesion impact on head vitality [7–11]. Vascular input to the femoral head is threatened by fracture since the epiphysis and most of the neck is intracapsular. Trueta and Harrison [7] provide the reference here, completing the work of Howe et al. [8] and Judet et al. [9]. In an anatomic study of 15 injected samples, Trueta and Harrison [7] determined the essential vascular elements of the femoral head as being the retinacular vessels originating from the medial circumflex artery; intra-osseous cervical vessels play a minor role and those originating downstream of the neck do not feed anything more than the lateral quarter of the head. These findings were confirmed by Sevitt and Thompson [10]. The lateral circumflex artery supplies few anterior retinacular vessels, so that lesions to it, whether traumatic or iatrogenic, have limited impact. The main extra-osseous arteries are the retinacular arteries deriving from the medial circumflex artery, which in turn is usually a branch of the deep femoral artery [11]. The medial circumflex artery is extra-capsular, with branches penetrating the capsule to become the inferior, posterior and superior retinacular vessels (Figs. 1 and 2).

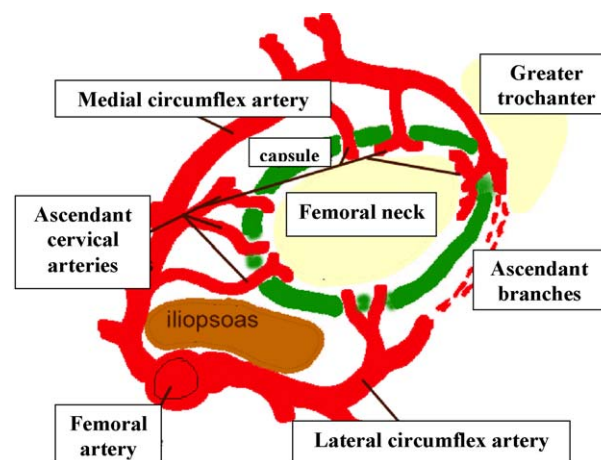


Figure 1 Vascularization of the superior extremity of the femur. Horizontal cross-section through the femoral neck and greater trochanter, left side.

Retinacular vessels

The retinacular arteries lie medially to the femoral head (Fig. 3), and mainly comprise the superior and inferior retinacular vessels. The anterior and posterior vessels are of less importance. Taken together, these arteries form Hunter's arterial circle [12].

There are between four and six superior retinacular vessels [7], which have the largest diameters, at a mean 0.8 mm. They penetrate the head at the superomedial and terminal part of the neck, at the cervicocephalic junction, to form the cervical branches: the superior metaphyseal and lateral epiphyseal arteries, of which the latter is the main artery, feeding 70 to 80% of the femoral head [7,10]. It runs along the old growth plate, anastomosing with the ligament teres vessels, and lies near the retinacular reflection area of the femoral neck. When these vessels are intact, the entire femoral head as well as the fovea can be injected via the anastomoses in cadaver studies. The inferior retinacular vessels are smaller and fewer (one or two) [7], with cervical branches penetrating the cortex near the anterior part of the head, finishing in an inferior metaphyseal branch in the inferior quarter of the head. They feed the distal metaphysis in two-thirds of cases. Sevitt and Thompson [10] conclude that these vessels are of little importance to head survival, despite the anastomosis network connecting them to the superior retinacular vessels.

Ligament teres artery

The ligament teres artery branches from the obturator artery, and anastomoses with the circumflex vessels, enabling injection via the common femoral artery. It crosses the head in a variable manner but, when it passes the fovea, it anastomoses with the terminal branches of lateral epiphyseal artery [7]. The role played by this anastomosis would seem from the literature to be controversial. Sevitt and Thompson [10] do not consider the ligament teres vessels to be essential to the survival of the head: after sectioning the neck, only a small part of the fovea was injected in two-thirds of their anatomic specimens, feeding only a very small

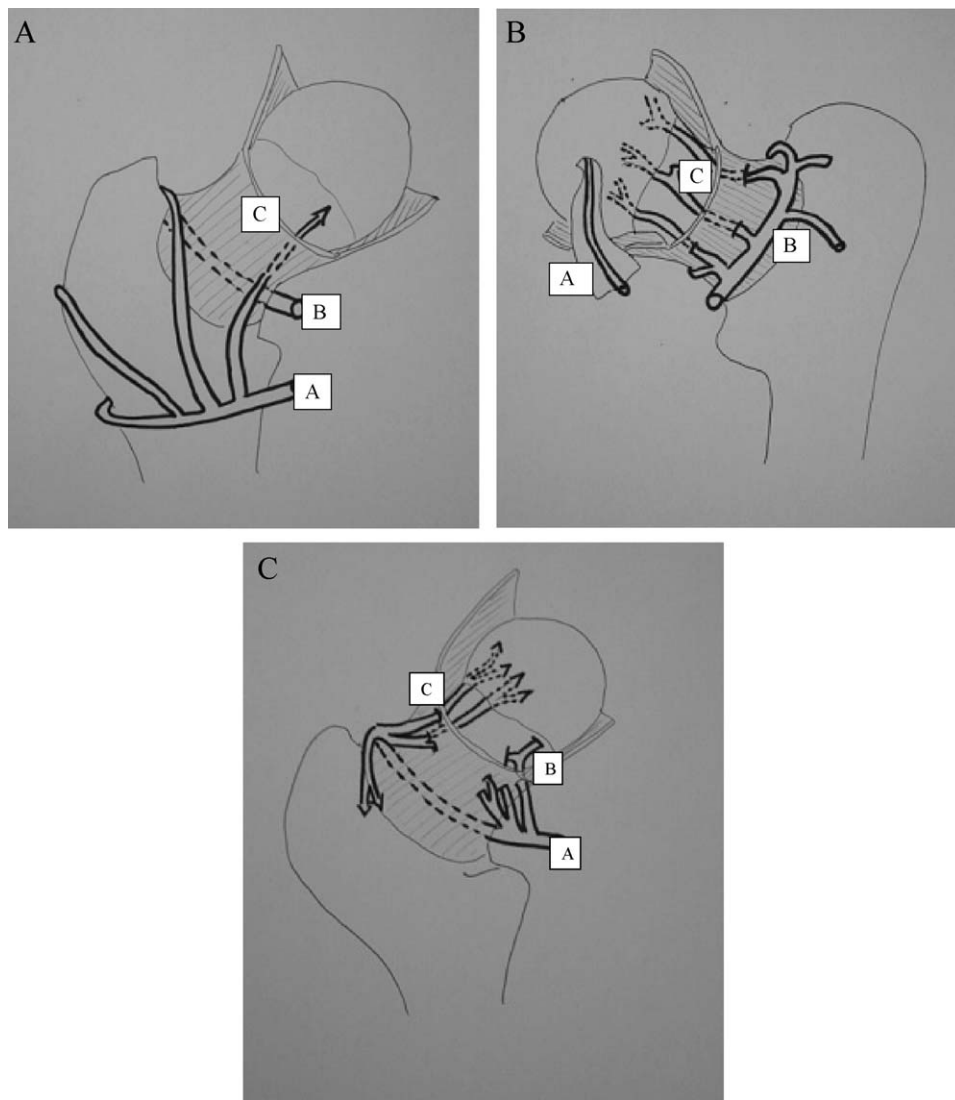


Figure 2 Vascularization of the superior extremity of the femur, right side. A. Anterior view: A. Lateral circumflex artery; B. Medial circumflex artery; C. Anterior nutrient branch. B. Posterior view: A. Medial circumflex artery; B. Ligament teres artery; C. Retinacular arteries. C. Anterior transparency view of medial circumflex artery: A. Medial circumflex artery; B. Inferior retinacular arteries; C. Superior retinacular arteries.

part of the head. Catto [13], Chandler and Kreuscher [14] and Crock [15], on the other hand, consider the anastomosis system between the ligament teres and lateral epiphyseal arteries to be essential to revascularization of the femoral head after neck fracture.

Physiopathology and natural history of post-traumatic necrosis

Functional prognosis in femoral neck fracture managed by osteosynthesis is largely dependent on the occurrence of head necrosis. The neck fracture causes vascular lesions, the devastating consequences of which have been demonstrated in several studies [2,16,17]. Sevitt [18] reported 84% of femoral heads to show devascularization, in a series of 25 anatomic specimens from neck fracture. The degree of displacement has been shown to determine the occur-

rence of vascular lesions, although fractures with low-grade displacement may also induce ischemic damage. Hemodynamic studies [2,4,19,20] showed significantly disturbed vascularization, with reduced blood-flow up to 60% after low-displacement fractures [2].

During trauma, synovial and retinacular vessels are detached or ruptured and intra-osseous vessels are broken by the fracture line. Damage to retinacular vessels upstream of the fracture is proportional to displacement and posterior comminution, a severity factor in terms of treatment, due to the ensuing difficulty of fixation and reduction, and in vascular terms. The lateral epiphyseal (extra-osseous) artery and posterior retinacular (extra-osseous) vessels are broken when the fracture line extends to the periphery of the femoral head. Retinacular artery and in particular lateral epiphyseal artery lesions almost systematically induce necrosis and are a fundamental element in subsequent collapse of the head [18]. Venous lesions (rupture and

thrombosis) also contribute to the necrotic process. Superior retinacular vessels rupture leaves the femoral head dependent on ligament teres and inferior retinacular vessels [10]. However, displacement may be so severe as to induce lesions in the latter, leaving only the ligament teres vessels to revascularize the head [13]. Sevitt [18], however, considers the ligament teres artery to be insufficient for complete revascularization.

As well as direct vascular lesions, there are other factors of poor prognosis. In low-grade displacement, capsule integrity causes the hematoma to be concentrated, increasing pressure and creating a tamponade effect [4]. Increased intracapsular pressure has several effects. The first is venule and arteriole thrombosis [4,21], followed by reduced blood-flow, inducing ischemic cellular necrosis [19,20]. Normal intra-articular pressure ranges from 0 to 20 mmHg [22] and normal arteriole pressure from 40 to 80 mmHg [4]. This baseline arteriole pressure, like that of muscle capillaries in leg compartment syndrome, accounts for the tissue tolerance observed up to an intra-articular pressure of 80 mmHg [23]. Holmberg and Dalen [4] demonstrated increased intra-articular pressure often exceeding 80 mmHg in case of fracture without displacement: in a series of nine non-displaced femoral neck fractures, all cases with grater than 80 mmHg pressure were abnormal on scintigraphy [4]. The position of the hip also affects intra-articular pressure: the association of extension, internal rotation and abduction reduces joint-space volume and increases the intracapsular pressure [4,22]; hip flexion has the opposite effect, reducing pressure by enlarging capsule volume. Anterior relief capsulotomy may limit the necrosis risk when intracapsular pressure exceeds arteriole pressure [23,24]. It is important to bear in mind that, in case of femoral neck fracture, excessive internal rotation and/or traction may aggravate vascular lesions by increasing the intracapsular pressure. The risks of surgery are thus non-negligible, at both instal-

lation and fixation, with a risk of head fragment rotation. Specific adapted management is required [1]. The interval to surgery is certainly an essential factor, influencing ischemia time in the head [1], but the quality of reduction is fundamental. Anatomic reduction has been clearly shown to reduce the risk of necrosis [25–27]. Revascularization quality is the key factor, especially as retinacular vessel integrity may be conserved in certain fractures [28]. Reduction should be performed quickly and perfectly, to optimize subsequent vascular reperfusion [29].

Biology of post-traumatic osteonecrosis of the femoral head

In post-traumatic necrosis, cellular proliferation quickly invades the femoral head, inducing significant osteogenesis. The “pathologic fracture”, responsible for the collapse of the femoral head, occurs at the interface between the new and the necrotic bone. Head deformity is caused not by cell death but by the repair process. Glimcher and Kenzora [30–33] performed the reference studies in this regard, demonstrating the secondary nature and vascular origin of head deformity. There are two stages in necrotic bone repair:

- cellular proliferation with invasion of the head by repair tissue;
- mesenchymatous cell differentiation into osteoblasts forming bone tissue on the surface of dead trabeculae.

In post-traumatic necrosis, once repair tissue has crossed the subcapital fracture line, it extends to form new bone. Arrest of osteoblast differentiation and of osteogenesis is related to intra-head microfractures blocking the process by inducing mesenchymatous differentiation into fibroblasts forming a fibrous layer similar to that found in non-union. The development of this fibrous tissue and the mobility of the fragments lead to resorption of the necrotic trabeculae. The direct cause of the changes undergone by the femoral head is thus not cell death but rather the action of the living cells involved in the repair process, altering the mechanical properties and inducing collapse of the head. The starting point of the collapse is the area of least resistance on the lateral side of the head, created by subchondral bone resorption secondary to the repair process. Collapse shifts towards the center, at the interface between the necrotic cancellous bone and the living bone, due to differences in consistency and elasticity between the two. This 2-stage theory of necrosis was confirmed by Steib et al. [34]: intra-osseous vascular assessment of necrotic hips using radioactive microspheres found no correlation between macroscopic arterial anatomy and functional vascular anatomy; they highlighted the importance of intra-osseous communication and the fundamental role played by the lateral epiphyseal artery [34].

Residual head perfusion assessment methods

Numerous methods of early assessment of residual femoral head perfusion following neck fracture seek to iden-

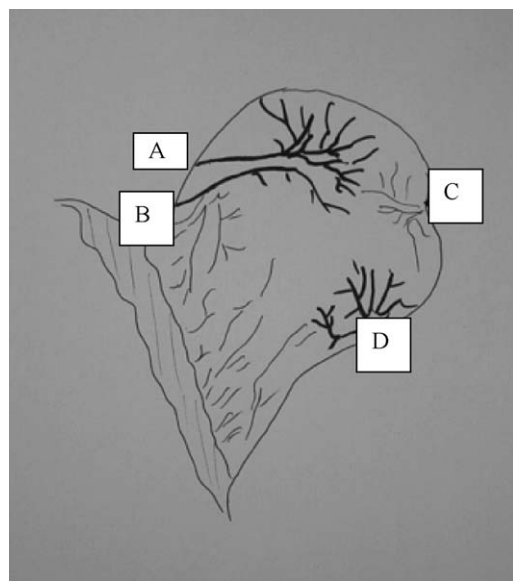


Figure 3 Head microcirculation according to Trueta and Harrison [7]. A. Lateral epiphyseal artery. B. Medial epiphyseal artery. C. Superior metaphyseal artery. D. Inferior metaphyseal artery.

tify subjects at risk of osteonecrosis. Some are invasive (superselective angiography, intra-osseous oxygen pressure measurement, or Doppler-laser hemodynamic measurement), while others involve imaging (scintigraphy, or classical or dynamic MRI).

Superselective angiography

Superselective angiography (SSA) analyzes extra-osseous arterial structures and was first used, in 1977, by Thérion [35] to assess head vascularity, which he showed could only be analyzed by catheterizing the medial circumflex artery so as to opacify the superior capsular vessels. These can be visualized even more effectively by digital subtraction arteriography (DSA), whereby Heuck and Raiser [36] highlighted vascular changes in nearly 97% of cases of confirmed post-traumatic necrosis, showing vascular lesions to be the main etiological factor. Langer et al. [37] applied DSA in nine patients presenting with recent femoral neck fracture and found eight interruptions or complete thromboses of superior nutrient branches originating in the medial circumflex artery. Taking all etiologies of necrosis together, they found superior medial circumflex artery branch involvement in 66% of cases; they recommend this technique for assessing necrosis risk and following up osteonecrosis managed by vascularized graft. Angiography is of undoubted interest for direct visualization of the vascular lesions underlying osteonecrosis, but has certain drawbacks, such as its invasive nature, entailing a risk of complications (arterial dissection and thrombosis, and hematoma), and especially the degree of expertise required to perform such a specialized examination. It shows up lesions at the time of fracture and lesions observed in confirmed necrosis; no studies, however, have found any correlation between lesion type and subsequent osteonecrosis.

Intra-osseous oxygen pressure measurement

Watanabe et al. [38] recently reported on femoral head intra-osseous oxygen pressure measurement following fracture, using a surgically implanted subchondral polarographic electrode. The hypothesis was that subchondral ischemia contributed to the development of avascular necrosis, and the aim was to assess avascular necrosis risk. Measurements were performed peroperatively, the electrode directly implanted at two points in the femoral head, in 17 patients (18 fractures); follow-up used MRI. A correlation was found between necrosis (seven cases) and oxygen pressure values at the two points. A statistically significant difference in pressure (i.e., of 3 mmHg) between the two points was considered as indicating a risk of necrosis, whereas comparable readings between the two points confirmed head viability. The technique is of special interest in non-displaced fracture, where there is theoretically less risk of necrosis. The study was based on that of Kiaer et al. [39], who demonstrated reduced oxygen pressure in subchondral bone in pathological (arthritis or confirmed necrosis) hips. They found a correlation between intra-osseous pressure, which was elevated in hip necrosis, and oxygen pressure, which was reduced. (As a reminder,

intra-osseous pressure in the femoral head is normal at 24 mmHg [40] and oxygen pressure at greater than 60 mmHg [39].) This is an elegant method for assessing tissue distress, but is invasive and difficult to perform in everyday practice.

Doppler-laser hemodynamic measurement

Swiontkowski et al. [41] were the first to use Doppler-laser flowmetry in the assessment of confirmed osteonecrosis, comparing necrotic areas to a presumed healthy reference area (trochanteric region). Doppler laser measured blood-flow through an introducer implanted in the femoral head. Measurements were taken in the trochanteric region, femoral neck, periphery of the collapsed femoral head segment and subchondrally: blood-flow was found to be reduced in necrotic areas. Recently, Sugamoto et al. [42] applied this method in recent cervical fracture, correlating results to fracture displacement on the Garden scale: flow was elevated and sinusoidal in Garden I, II and in certain cases III, but low and non-sinusoidal in other cases of Garden III and all of Garden IV. Values were less than 10 perfusion units, and waves did not synchronize with heartbeat; the authors stressed that there is no severe vascular damage when there is synchronization with heartbeat and values exceed 30 perfusion units. Femoral head blood-flow following pertrochanteric fracture served as reference value, as such fractures are theoretically free of necrotic complication: the curves were considered normal, with sinusoid rhythm and elevated values. This technique resembles macroscopic analysis by angiography, but analyzes the femoral head with greater precision, like oxygen pressure measurement; it has, however the drawbacks of instrumentation cost and invasiveness.

Bone scintigraphy

Results on hemodynamic status are qualitative rather than quantitative. Scintigraphy provides earlier hemodynamic information in the vascular phase and metabolic information in the osseous phase than does radiology. The method is more sensitive and gives earlier results than computed tomography (CT), analyzing vascular perfusion and bone-marrow uptake on images taken during and after radio-tracer injection. The vascular changes precede onset of the necrotic process: imaging during the first 24 h reveals disturbed hemodynamics, with early histochemical abnormality.

Tucker [43] introduced the use of a radioactive tracer in the diagnosis of femoral osteonecrosis in 1950, and this was taken up again by Boyd et al. [44] in 1955. Using ^{32}P phosphorus, they measured the trochanter/head intensity ratio peroperatively on a Geiger-counter. The first report on scintigraphy in the diagnosis of femoral head necrosis was by Riggins et al. [45]. The rise of Technetium $^{99\text{m}}$ bone scintigraphy for assessment of residual vascularity dates back to the late 1970s, notably with Webber et al. [46] and Meyers et al. [47]. This isotope was chosen as being specific to tissue vascularity disturbance in general and of bone in particular, being phagocytosed by bone-marrow reticulocytes. Meyers et al. [47], in a prospective series of 95 neck frac-

tures, two-thirds with displacement, reported about 95% accuracy in the estimation of necrosis risk at 2 years' FU. They found the technique to be easy of access and relatively risk-free, and recommended early scintigraphy within the first 24 h of fracture to assess necrosis risk and to guide treatment. Philipps et al. [48] reported identical findings in a series of 30 recent fractures in which scintigraphy was performed within 72 h. D'Ambrosia et al. [49], using Technetium 99m diphosphonate to diagnose necrosis, found it to show good sensitivity, highlighting a hypofixation area in the early course, before any signs appeared on standard X-ray. In a "dynamic" prospective study, Bauer et al. [50] assessed neck fracture evolution, performing Technetium 99m-methyldiphosphonate scintigraphy postoperatively and at 4, 8 and 12 months: isotope activity fell off over time, comparing the fractured and healthy sides. Lucie et al. [51] analyzed 92 recent fractures on Technetium 99m phosphate scintigraphy: accuracy was 91% in the "normal scintigraphy" group and 82% in the "pathologic scintigraphy" group. Greiff et al. [52,53] reported similar findings with the same isotope in early assessment of femoral head vitality after fracture and also in diagnosis of confirmed necrosis. Finally, Turner [54] used technetium 99m antimony colloid scintigraphy in an experimental and clinical study in the first 24 h after neck fracture in 30 patients; 16 fractures were normal and 14 pathological; 13 of the latter developed necrosis within 2 years; accuracy was 87%.

It is established that scintigraphy is positive some 14 months before any radiological signs [52]. The present review of the literature suggests that the latest generation of scintigraphy provides a reliable examination, with 85 to 90% accuracy in the assessment of residual femoral head hemodynamics following recent neck fracture; it is also more accessible and cheaper, but with the iatrogenic risks which, however rare, are inherent to the use of isotopes. It does not, however, provide morphological analysis of the superior extremity of the femur, and requires considerable expertise in interpretation.

Magnetic resonance imaging

MRI is the reference examination for the diagnosis of avascular necrosis of the femoral head. Lang et al. [55] report 90–100% sensitivity and 100% specificity. Many publications, going back to the 1980s, have shown its potential in symptomatic patients [56,57]. More recent studies have highlighted the interest of MRI for early diagnosis of asymptomatic necrosis [58]. Basset et al. [59] showed that MRI provided earlier diagnosis than scintigraphy. Various authors therefore sought to determine a minimum interval for positive MRI in osteonecrosis: reports are not unanimous, with intervals ranging from a few days to several months. Nakamura et al. [60], in a dog model of neck fracture, found a minimum interval of 7 days for the first signs of osteonecrosis to appear in certain animals but an interval of 4 weeks for all 25 of the dogs. Osteonecrosis sets in three to five times as early in dogs as in humans, so that the authors estimate the minimum interval to osteonecrosis detection on MRI to be some 4 weeks. Several human studies have been performed. In a series of 15 neck fractures, Speer et al. [61] reported a minimum interval of 48 hours to detect signs of osteonecrosis

on MRI. In a series of 28 hip fractures analyzed on radiology and histology, Asnis et al. [62] found that MRI detected osteonecrosis as of 2 weeks. Finally, two studies focused on the follow-up of fractures managed by osteosynthesis, to determine the optimal interval to diagnosis of osteonecrosis [63,64]. Kawasaki et al. [64] followed up 31 fractures for 12 months, performing MRI at 2, 6 and 12 months: signs of osteonecrosis appeared in 39% of cases by 6 months, and the authors concluded that a 6-month interval provided the best sensitivity, specificity and accuracy in diagnosing (present) post-traumatic osteonecrosis [64]. All of these studies were based on MRI sequences that did not allow assessment of femoral head hemodynamics or of the very early cell impact of ischemia; bone-marrow signal variations mainly depend on fatty cells, which can survive without oxygen for 2 to 5 days, during which the fatty signal remains normal [61].

Interest then shifted to dynamic MRI with contrast medium injection. The first studies on animal models were published by Cova et al. [65] and Nadel et al. [66]. Lang et al. [67] reported on a series of 13 recent femoral neck fractures, correlating MRI results with superselective angiography (7 cases) or 12-months' radioclinical follow-up (six cases); they found MRI to be useful, with one limitation: poor physiological enhancement of bone-marrow in elderly subjects. Kamano et al. [68] reported on a series of 29 patients who underwent dynamic MRI within 24 hours of femoral neck fracture. Results were classified according to three levels of head enhancement: type 1 = no enhancement, type 2 = partial enhancement, type 3 = complete enhancement. On a mean MRI follow-up of 27 months, there was 100% necrosis in type 1, 50% in type 2, and none in type 3. The authors found the technique to be very reliable, with perfect prediction in types 1 and 3, and with the great advantage of being non-invasive. Konishiike et al. [69] confirmed these findings in a series of 22 fractures explored by dynamic MRI within 48 hours of fracture. They distinguished three types of results, comparing enhancement curves between the fractured and healthy sides: type A = identical curves, type C = flat fracture-side curve, type B = intermediate fracture-side aspect. Type-B curves were interpreted as an intermediate hemodynamic state not systematically requiring arthroplasty. In a control group of pertrochanteric fracture, all curves were type A. The same team later reported on a series of 36 fractures with a minimum 2 years' FU [70]. Fractures showing an initial type-C curve were followed up on MRI to determine the predictive value of initial dynamic MRI for subsequent osteonecrosis risk. Sensitivity was 80% and specificity 100%: i.e., 89% accuracy. The authors concluded that their new MRI classification could guide indications for surgery: in their current practice, conservative treatment is indicated in case of type A or B curves. Kubo et al. [71] reported identical results using color mapping (red = normal perfusion, black = no perfusion) on dynamic images. Three types were distinguished: type A = red, identical to the healthy side, type B = intermediate, and type C = black, with total absence of perfusion. This classification is of immediate impact on choice of treatment. Finally, an Indian team recently once again demonstrated the interest of dynamic MRI [72].

These dynamic MRI studies are especially interesting, showing that certain non-displaced fractures are associ-

ated with head vascularity disturbance while in other cases enhancement patterns may be normal despite severe displacement. This is a genuinely new classification, which may be said to be “functional”, based on early assessment of residual head vascularization after neck fracture, independently of anatomic fracture type. The MRIs were performed within 48 hours of fracture, but such an interval represents a lost opportunity for head revascularization secondary to early reduction and stabilization: ideally, they should be performed in the first 10 hours after fracture so as not only to assess necrosis risk but also give the patient the best possible chance. MRI availability is another issue, and an important limitation.

Revascularization assessment

Once the fracture has happened, the vascular assessment has been made and treatment has been performed, there is a technique that enables the revascularization and cicatrization process to be monitored, which may also be highly relevant to patient follow-up. Phlebography by direct intracephalic puncture was recommended by Jenny and Vecsei [73] for the follow-up of fixed neck fracture. Weight bearing was authorized only after consolidation and more importantly after confirmation of recovery of head vascularity. The basic principle was that head revascularization is enhanced by early high-quality reduction, stable synthesis under compression and complete rest for the hip, as classically agreed in the literature [1]. The authors consider the hip to be normal, with restored vascularity, once the phlebographic assessment shows efferent vessel opacification and complete vascular draining.

Present and future strategy

The literature testifies to disturbance of femoral head vascularity after neck fracture, preparing the ground for avascular necrosis. A less common concept is that of hypohemia of the head, which highlights head fragility even more. Nakamura et al. [74] used positron emission tomography (PET) to demonstrate that the femoral head exhibits permanent hypohemia compared to neighboring bone tissue (trochanteric region, ilium), with lower blood volume and flow. This seems to be an important notion, reinforcing the legitimacy of the initial post-traumatic vascular assessment, which may be worth extending contralaterally to enable comparison and weighting of results.

The literature contains reports on various techniques of residual head vascularity assessment that have proved their efficacy. Routine application, however, in some cases (oxygen pressure measurement, or Doppler) raises both economic and practical issues. SSA and DSA cannot presently be used systematically due to attendant iatrogenic risk and because other imaging techniques are more effective and enable earlier analysis while being less invasive. Choice favors scintigraphy and MRI. In the light of the literature, scintigraphy appears to be both effective and highly sensitive. The patient is, however, exposed to radiation in the form of the injected radioactive isotope. Moreover, scintigraphy does not provide morphometric analysis and requires expertise on the part of the nuclear physician. MRI is the

great hope, looking at the literature, with surprising results that have stirred debate that may yet change therapeutic practice. Dynamic MRI seems to be even more promising, and represents the future for early assessment of femoral head necrosis risk. As we have seen, several teams have demonstrated its efficacy and developed functional classifications for femoral neck fracture that are not bound by classical anatomic observation criteria. This is a real revolution in the world of traumatology, with treatment indications that are independent of fracture morphology and based rather on criteria of residual vascularity and hence on risk of ischemia. The main limitation is one of availability of equipment and of expertise. It is clear that dynamic MRI performed within 8 hours of neck fracture would optimize management of young patients. MRI should not, however, delay conservative surgery by osteosynthesis, which we consider should be performed in emergency within a maximum 10 to 12 hours as the best guarantee of femoral head survival. This is not the place to go into issues of procedure; suffice it to say that assembly should follow certain ground-rules: stability and anatomic reduction, with anterior arthrotomy on a Watson-Jones approach, if required, to control reduction and especially head fragment rotation, and postoperative non-weight-bearing as a safety measure [1].

This “diagnostic therapeutic” approach using early dynamic vascular imaging should be the target, however difficult to implement in practice. The various reports of osteosynthesis in fracture [68–72] highlight the interest of the technique as suggesting a treatment algorithm, without actually recommending one. In young patients (under 65 years of age) [1], management should be conservative, whatever the type of result. The result enables necrosis risk to be estimated from the initial assessment and above all allows the patient to be informed of the degree of risk. This patient information is important, in a society of transparency and dialog. In elderly patients (over 65 years of age), some authors recommend conservative management [68–72] in case of “normal” vascularity, and replacement where vascularity is considered to be lacking, whatever the “morphologic” type of the fracture. If a fracture is stable, and therefore relatively free of displacement, yet exhibiting poor vascular status, replacement is to be preferred over the conservative treatment that would classically have been recommended. It is in this sense that it is appropriate to talk of “functional” classification. It is finally in these situations that vascular assessment comes into its own. Thus, in situations of intermediate vascularity, age should be the determining factor in guiding management: calendar age, obviously, with an 80-year threshold beyond which hip replacement is to be recommended, but also physiological age [1]. For a given calendar age (of between 65 and 80 years), a patient may either be “physiologically young” or “physiologically fatigued”, depending notably on associated visceral defects and comorbidities. The essential factor, which in our view enables classification, is life-expectancy [1], estimated firstly by judging the patient’s degree of independence/dependence in terms of place of residence (home, alone or accompanied, institution), walking status (cane, frame or need of help) with or without walking activity, and ability to perform everyday activities and tasks (shopping, washing, house-

work). All of this also sheds indirect light on mental status, for which we use Parker's score [1]. Finally, assessment of autonomy is weighted by the patient's history. Thus, a patient deemed "physiologically young", who lives alone at home, walks without assistance or just with a cane to go outdoors, performs everyday activities independently and has no particular history, will have a life-expectancy estimated at more than 10 years. Conversely, a patient who systematically uses a walking aid, lives in an institution, is dependent for everyday activity or has a heavy history will be deemed "physiologically fatigued" with a shorter life-expectancy. Conservative treatment may be recommended in the former case, specifying the situation and attendant risk; in the latter case, hip replacement is to be recommended.

This literature review has clearly shown the interest of early initial functional vascular assessment enabled by dynamic MRI. Unfortunately, availability (in number, and in emergency settings) and expertise set a limit. Lack of MRI equipment, especially in emergency settings, in France is fundamental and, in our opinion, constitutes a real problem of public health. We therefore wondered whether this vascular information might not be harvested in some easier manner. We sought to meet two criteria: perfusion assessment, and ease of access to the examination. For this, we turned to CT perfusion scan, which combines technical simplicity with ready availability of equipment. An evaluation of the efficacy of CT perfusion in necrosis risk assessment by analysis of post-traumatic residual head vascularity is currently underway.

Conflicts of interest statement

None.

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